The Sixth Sense

In his article, "Neurogenic Inflammation and Sensitivity to Environmental Chemicals" (101:234-238), Meggs speculates that neuropeptide mediators of inflammation may be responsible for sick building syndrome and multiple chemical sensitivity syndrome (MCS). He invokes a novel sixth sense, the "common chemical sense," in the noses of such patients. Meggs's speculations greatly exceed the scientific knowledge of the functions of neuropeptides. He seems to accept that these clinical conditions actually exist, although he acknowledges that MCS "remains highly controversial." In fact, there is a substantial body of literature (1-9) that these syndromes are largely psychogenic and are exploited by "alternative medicine" practitioners and quacks who ignore the distinction between speculation like Meggs's and proven fact, and who disregard the need for proof before clinical implementation. The American Medical Association Council on Scientific Affairs believes that multiple chemical sensitivity should not be considered a recognized clinical syndrome (10).

We appreciate Meggs's call for more scientific research into these matters. In the meantime, we hope he is not providing more grist for the quacks' mill.

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REFERENCES

- American College of Physicians. Position paper: clinical ecology. Ann Intern Med 111:168–178 (1989).
- Black DW, Rathe A, Goldstein RB. Environmental illness: a controlled study of 26 subjects with "20th century disease." J Am Med Assoc 264:3166(1990).
- Brodsky CM. "Allergic to everything": a medical subculture. Psychosomatics 24:731(1983).
- Cullen MR, ed. Workers with multiple chemical sensitivities. State Art Rev Occup Med 2(1987).
- Kahn E, Letz G. Clinical ecology: environmental medicine or unsubstantiated theory? Ann Intern Med 111:104–106(1989).
- Pearson DJ. Psychologic and somatic interrelationships in allergy and pseudoallergy. J Allergy Clin Immunol 81:351(1988).
- Stewart DE, Raskin J. Psychiatric assessment of patients with "20th century disease" (total allergy syndrome). Can Med Assoc J 133: 1001(1985).
- Terr A. Environmental illness. A clinical review of fifty cases. Arch Int Med 146:145–149 (1986).

- 9. Terr A. Clinical ecology. J Allergy Clin Immunol 79:423–426(1987).
- Council on Scientific Affairs, American Medical Association. Clinical ecology. J Am Med Assoc 268:3465-3467(1992).

Pesticides on Food

On page 390 of the October issue of EHP (volume 101, number 5), there is a graph that purports to show the intake of pesticides by children in milligrams per kilogram per day. I think you owe a prominent correction/explanation to your readers. The original figure in the National Academy of Sciences report (figure 5-1, p. 172) shows that this is intake of food, not pesticide residue. The only point of the figure is that infants eat more of certain commodities than do adults on a body weight basis. If there is residue present, and if it survives processing, then they would get a correspondingly higher exposure. However, the situation is nothing like you imply.

Furthermore, I could not find any place in the report that says children consume 60 times more fruit than adults. Table 5-5 of the NAS report (p. 183) shows that apple juice may be consumed by non-nursing infants at 16 times the national average (again, relative to body weight). For 1-6 year olds, the factor is 3 or less for most all foods.

Finally, while it is true that concern about this report generated much activity within Congress and several federal agencies, a careful reading of the actual report will show that the concerns are largely theoretical in nature. Improvements are desired in consumption data, toxicity testing, overall regulatory approach, etc. There is nothing in the report, despite quotes to the contrary, that demonstrates that the food supply is unsafe for children or any other subset of the population.

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Editor's Note: The caption for the graph on page 390 of volume 101 refers to infant intake of pesticides on raw agricultural commodities, but the graph shows only amounts of food intake in proportion to body weight, not pesticide residues. We regret any confusion resulting from this caption.

Heterocyclic Amine-induced Cancer and Myocardial Lesions in Nonhuman Primates

Two articles in this issue from Adamson and co-workers (p. 190) describe the effects of feeding monkeys carcinogens formed during the cooking of food derived from animal muscle. In the first paper the frequency and descriptive pathology is reported for 2-amino-3-methylimidazo[4,5-f]quinoline (IQ)-induced liver tumors. In the second paper, a histopathological study of perfusion-fixed hearts of tumor-bearing monkeys showed a variety of myocardial lesions with exposure to IQ.

The major impact of this work is that a nonhuman primate species, the cynomolgus monkey, develops liver tumors after exposure to a heterocyclic amine that is ubiquitous in our cooked food supply (1,2). Not only do the monkeys under test get tumors, but 43 months was the average latent period for the high-dose animals and is equivalent to 15–25% of the animals' life span—a very quick response.

An important question arises from this research: Do the high doses (10 and 20 mg/kg) used chronically in these experiments relate to human exposures, and if not, are the results still significant? Humans eating well-done muscle-derived meats consume 10,000-100,000 times less material daily per kilogram of body weight than do the monkeys (3). There are a number of studies that attempt to answer this question about high-dose extrapolation. They suggest that at 104-106 times lower doses than used in the feeding studies discussed here, heterocyclic amines survive the acid in the stomach, are taken up by the bloodstream from the intestine, and are metabolized by the liver cytochrome P450-A metabolizing enzymes (4,5). The N-hydroxy metabolites are then either reactive in the liver after further conjugation to form DNA adducts and presumably liver tumors or are found as DNA adducts in numerous nonhepatic tissues where the conjugation reactions probably occur locally (6). The question then is do these reactions happen when the reactant is at 10,000 times lower concentration? Apparently, the answer is yes. In specific rodent experiments conducted over many orders of magnitude of dose, DNA binding for these compounds appears linear down to the levels found in a single hamburger (7). The data suggests that repair of DNA damage (heterocyclic amine adducts)